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Total (1)	27	-	-

once an inventorship error is discovered, timeliness requirements under 37 CFR 1.116 and 37 CFR 1.312 apply. For allowed applications where the issue fee has been paid prior to the entry of a request under 37 CFR 1.48, if the request under 37 CFR 1.48 is dismissed or denied in an Office action, the application must be withdrawn from issue so that applicant would be given time to correct the defect(s). If the request under 37 CFR 1.48 is granted, then it would not be necessary to withdraw the application from issue.

Requests under 37 CFR 1.48 are generally decided by the primary examiner except:

(A) When the application is involved in an interference (decided by the Board of Patent Appeals and Interferences);

(B) When the application is a national stage application filed under 35 U.S.C. 371 which, as of the date of filing of the request, has not been accepted as satisfying the requirements for entry into the national stage (decided in the PCT Legal Office); >and<

(C) When accompanied by a petition under 37 CFR 1.183 requesting waiver of a requirement under 37 CFR 1.48(a) or (c), e.g., waiver of the statement of lack of deceptive intent by an inventor to be added or deleted, or waiver of the reexecution of the declaration by all of the inventors (decided in the Office of Petitions). **

When any request for correction of inventorship under 37 CFR 1.48(a)-(c) is granted, the examiner will acknowledge any addition or deletion of the names of inventors by using either form paragraph 2.14 or form paragraph 2.14.01 in the next Office communication to applicant or his/her attorney. It will be necessary to revise the PALM records, issue a corrected filing receipt, and change the bib-data sheet on the file wrapper. The correction should be noted on the original oath or declaration by writing in red ink in the left column "See Paper No. __ for inventorship corrections." See MPEP § 605.04(g).

¶ *2.14 Correction of Inventorship Under 37 CFR 1.48(a) or (c), Sufficient*

In view of the papers filed [**1**], it has been found that this nonprovisional application, as filed, through error and without deceptive intent, improperly set forth the inventorship, and accordingly, this application has been corrected in compliance with 37 CFR 1.48 ([**2**]). The inventorship of this application has been changed by **[3]**.

The application will be forwarded to the Office of Initial Patent Examination (OIPE) for issuance of a corrected filing receipt, and correction of the file jacket and PTO PALM data to reflect the inventorship as corrected.

Examiner Note

1.

In bracket 2, insert --a-- or --c--, as appropriate.

2.

In bracket 3, insert explanation of correction made, including addition or deletion of appropriate names.

¶ *2.14.01 Correction of Inventorship Under 37 CFR 1.48(b), Sufficient*

In view of the papers filed [**1**], the inventorship of this nonprovisional application has been changed by the deletion of [**2**].

The application will be forwarded to the Office of Initial Patent Examination (OIPE) for issuance of a corrected filing receipt, and correction of the file jacket and USPTO PALM data to reflect the inventorship as corrected.

Examiner Note

1.

change;

(2) A statement from each person being added as an inventor and from each person being deleted as an inventor that the error in inventorship occurred without deceptive intention on his or her part;

(3) An oath or declaration by the actual inventor or inventors as required by § 1.63 or as permitted by §§ 1.42, 1.43 or § 1.47;

(4) The processing fee set forth in § 1.17(i); and

(5) If an assignment has been executed by any of the original named inventors, the written consent of the assignee (see § 3.73(b) of this chapter).

(b) *Nonprovisional application —fewer inventors due to amendment or cancellation of claims* . If the correct inventors are named in a nonprovisional application, and the prosecution of the nonprovisional application results in the amendment or cancellation of claims so that fewer than all of the currently named inventors are the actual inventors of the invention being claimed in the nonprovisional application, an amendment must be filed requesting deletion of the name or names of the person or persons who are not inventors of the invention being claimed. If the application is involved in an interference, the amendment must comply with the requirements of this section and must be accompanied by a motion under § 1.634. Amendment of the inventorship requires:

(1) A request, signed by a party set forth in § 1.33(b), to correct the inventorship that identifies the named inventor or inventor's being deleted and acknowledges that the inventor's invention is no longer being claimed in the nonprovisional application; and

(2) The processing fee set forth in § 1.17(i).

(c) *Nonprovisional application —inventors added for claims to previously unclaimed subject matter* . If a nonprovisional application discloses unclaimed subject matter by an inventor or inventors not named in the application, the application may be amended to add claims to the subject matter and name the correct inventors for the application. If the application is involved in an interference, the amendment must comply with the requirements of this section and must be accompanied by a motion under § 1.634. Amendment of the inventorship requires:

(1) A request to correct the inventorship that sets forth the desired inventorship change;

(2) A statement from each person being added as an inventor that the addition is necessitated by amendment of the claims and that the inventorship error occurred without deceptive intention on his or her part;

(3) An oath or declaration by the actual inventors as required by § 1.63 or as permitted by §§ 1.42, 1.43, or § 1.47;

(4) The processing fee set forth in § 1.17(i); and

(5) If an assignment has been executed by any of the original named inventors, the written consent of the assignee (see § 3.73(b) of this chapter).

(d) *Provisional application —adding omitted inventors* . If the name or names of an inventor or inventors were omitted in a provisional application through error without any deceptive intention on the part of the omitted inventor or inventors, the provisional application may be amended to add the name or names of the omitted inventor or inventors. Amendment of the inventorship requires:

Human chemokines, CKbeta4 and CKbeta10/ MCP - 4 .

AUTHOR: Li Haodong(a); Adams Mark; Lima Solange Hanschke; Alderson Ralph; Li Yuling; Parmelee David; White John; Applebaum Edward
AUTHOR ADDRESS: (a)18 Observation Ct., Fox Run 303, Germantown, MD, 20878** USA

JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1242 (3):pNo Pagination Jan. 16, 2001
MEDIUM: e-file
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Human chemokine polypeptides and **DNA** /(RNA) encoding such chemokine polypeptides and a procedure for producing such polypeptides by recombinant techniques is disclosed. Also disclosed are methods for utilizing such chemokine polypeptides for the treatment of leukemia, tumors, chronic infections, autoimmune disease, fibrotic disorders, wound healing and psoriasis. Antagonists against such chemokine polypeptides and their use as a therapeutic to treat rheumatoid arthritis, autoimmune and chronic inflammatory and infective diseases, allergic reactions, prostaglandin-independent fever and bone marrow failure are also disclosed.

Human monocyte chemoattractant protein (MCP)- 4 is a novel CC chemokine with activities on monocytes, eosinophils, and basophils induced in allergic and nonallergic inflammation that signals through the CC chemokine receptors (CCR)-2 and -3.

Garcia-Zepeda E A; Combadiere C; Rothenberg M E; Sarafi M N; Lavigne F; Hamid Q; Murphy P M; Luster A D

Infectious Disease Unit, Massachusetts General Hospital, and Harvard Medical School, Charlestown 02129, USA.

Journal of immunology (Baltimore, Md. : 1950) (UNITED STATES) Dec 15 1996, 157 (12) p5613-26, ISSN 0022-1767 Journal Code: 2985117R

Contract/Grant No.: R01 CA69212-01; CA; NCI

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Monocyte chemotactic protein 4 (MCP - 4), a novel structural and functional analogue of MCP-3 and eotaxin.

Uguccioni M; Loetscher P; Forssmann U; Dewald B; Li H; Lima S H; Li Y; Kreider B; Garotta G; Thelen M; Baggiolini M

Theodor Kocher Institute, University of Bern, Switzerland.

Journal of experimental medicine (UNITED STATES) May 1 1996, 183 (5) p2379-84, ISSN 0022-1007 Journal Code: 2985109R

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

A novel human CC chemokine complementary **DNA** was identified in a library constructed from human fetal RNA, cloned into a baculovirus **vector** , and expressed in Sf9 insect cells. The mature recombinant protein that was released had the NH2-terminal sequence pyro-QPDALNVPSTC...and consisted of 75 amino acids. Minor amounts of two variants of 77 and 82 residues (NH2 termini: LAQPDA...and FNPQGLAQPDA...) were released as well. The novel chemokine was designated monocyte chemotactic protein 4 (**MCP - 4**) and the variants were designated (LA) **MCP - 4** and (FNPQGLA) **MCP - 4** . **MCP - 4** shares the pyroglutamic acidproline NH2-terminal motif and 56-61% sequence identity with the three known monocyte chemotactic proteins and is 60% identical to eotaxin. It has marked functional similarities to MCP-3 and eotaxin. Like MCP-3, **MCP - 4** is a chemoattractant of high efficacy for monocytes and T lymphocytes. On these cells, it binds to receptors that recognize MCP-1, MCP-3, and RANTES. On eosinophils, **MCP - 4** has similar efficacy and potency as MCP-3, RANTES, and cotaxin. It shares receptors with eotaxin and shows full cross-desensitization with this cosinophil-selective chemokine. Of the two variants, only (LA) **MCP - 4** could be purified in sufficient quantities for testing and was found to be at least 30-fold less potent than **MCP - 4** itself. This suggests that the 75-residue form with the characterist**9/3,AB/22 (Item 4 from file: 73)**

DIALOG(R)File 73:EMBASE

06911833 EMBASE No: 1997196275

Cloning, in vitro expression, and functional characterization of a novel human CC chemokine of the monocyte chemotactic protein (MCP) family (MCP - 4) that binds and signals through the CC chemokine receptor 2B

Berkhout T.A.; Sarau H.M.; Moores K.; White J.R.; Elshourbagy N.; Appelbaum E.; Reape T.J.; Brawner M.; Makwana J.; Foley J.J.; Schmidt D.B.; Imburgia C.; McNulty D.; Matthews J.; O'Donnell K.; O'Shannessy D.; Scott M.; Groot P.H.E.; Macphee C.

P.H.E. Groot, Dept. of Vascular Biology, SmithKline Beecham, Welwyn, Herts AL6 9AR United Kingdom

Journal of Biological Chemistry (J. BIOL. CHEM.) (United States) 1997 , 272/26 (16404-16413)

CODEN: JBCHA ISSN: 0021-9258

DOCUMENT TYPE: Journal; Article

LANGUAGE: ENGLISH SUMM

L2 ANSWER 1 OF 10 CAPLUS COPYRIGHT 2002 ACS
TI Analysis of the Gene Expression Profiles of Immature versus Mature Bone Marrow-Derived Dendritic Cells Using DNA Arrays
SO Biochemical and Biophysical Research Communications (2002), 290(1), 66-72
CODEN: BBRCA9; ISSN: 0006-291X
AB Dendritic cells (DCs) are professional antigen-presenting cells of the immune system and can be generated in vitro from bone-marrow cells. In this study, we systematically investigated by DNA array anal. the expression profiles of 514 immunol. relevant genes in two populations of mouse bone marrow-derived DC, immature (DCIMAT), and lipopolysaccharide (LPS)-stimulated mature (DCMAT) DCs. Our data showed that DCIMAT expressed transcripts for 69 (13.42% of the 514) of these genes and that, upon maturation, 32 (6.23%) of these were up-regulated and 40 (7.78%) down-regulated. Maturation-dependent up-regulation, defined by a differential expression (DE) ratio of >2, was obsd. among five cytokine (Flt-3L, TNF-.alpha., IL-1.alpha. and -1.beta., and IL-6), three chemokine (RANTES, MIP-2 and GROa) and three other (iNOS, MMP-13, and STRAP) genes. Reciprocally, maturation-dependent down-regulation occurred with one cytokine (IGF-1), two chemokine receptor (CCR2 and CCR5), and three other (RP105, Axl, and UCP2) genes. Lower level, but nevertheless significantly enhanced expression of the chemokine receptor CCR7 and of NF-.kappa.B was also obsd. upon DC maturation. This DC maturation profile confirms previous findings from other lab, but it also substantially broadens our view of these cells by documenting expression changes among genes (e.g., IGF-1, MMP-13, STRAP) not reported previously in these cells. (c) 2002 Academic Press.
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**RP-105**; gene expression profiles of immature vs. mature bone marrow-derived dendritic cells using DNA arrays)

L2 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2002 ACS
TI Chemokines as adjuvants of immune response
SO Eur. Pat. Appl., 16 pp.
CODEN: EPXXDW
AB Dendritic cells play a crit. role in antigen-specific immune responses. Materials and methods are provided for treating disease states, including cancer and autoimmune disease, by facilitating or inhibiting the migration or activation of antigen-presenting dendritic cells. In particular, chemokines are used to initiate, amplify or modulate an immune response. In one embodiment, chemokines are used to attract dendritic cells to the site of antigen delivery. An increase no. of dendritic at the site of antigen delivery means more antigen uptake and a modified immune response.
IT Cytokines
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**RP-105**; chemokines as adjuvants for inducing antigen-specific immune response)

L2 ANSWER 3 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
TI High performance liquid chromatographic determination of phenolic compounds in seed exudates of Festuca arundinacea and F. pratense.
SO Phytochemical Analysis, (November December, 2000) Vol. 11, No. 6, pp.

375-379. print.
ISSN: 0958-0344.
AB Derivatives of benzoic and cinnamic (caffeic, protocatechuic, vanillic, ferulic, p-coumaric, p-hydroxybenzoic and syringic) acids and 3,4-dihydroxybenzaldehyde in seed exudates of Festuca arundinacea cv. "Kora" and F. pratense cv. "Otava" have been separated and quantified on a Zorbax SB C18 Rapid ResolutionTM column with linear gradient elution using a mobile phase containing acetonitrile and 0.5% acetic acid. The USP tailing factors were less than 1.15 for all peaks. Strictly linear calibration curves (r > 0.9997) with limits of detection of 90-880 nM were obtained for all analytes in the concentration ranges from units or tenths up to 600 muM. **RP 105** and OASISTM HLB polymeric sorbents have been shown to be best of the tested sorbents for the separation and preconcentration of phenolic compounds from both plant materials. The solid-phase extraction procedures have been compared with classical liquid-liquid extractions and consistent results were obtained.
AB. . . of 90-880 nM were obtained for all analytes in the concentration ranges from units or tenths up to 600 muM. **RP 105** and OASISTM HLB polymeric sorbents have been shown to be best of the tested sorbents for the separation and preconcentration. . .

L2 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2002 ACS
TI The Toll-like receptor protein RP105 regulates lipopolysaccharide signaling in B cells
SO Journal of Experimental Medicine (2000), 192(1), 23-29
CODEN: JEMEAV; ISSN: 0022-1007
AB The susceptibility to infections induced by Gram-neg. bacteria is largely detd. by innate immune responses to bacteria cell wall lipopolysaccharide (LPS). The stimulation of B cells by LPS enhances their antigen-presenting capacity and is accompanied by B cell proliferation and secretion of large quantities of LPS-neutralizing antibodies. Similar to macrophages and neutrophils, the LPS-induced activation of B cells is dependent on Toll-like receptor (TLR)4. Here, we demonstrate that the responses of B cells to LPS are also regulated by another TLR protein, RP105, which is predominantly expressed on mature B cells in mice and humans. The anal. of mice homozygous for the null mutation in the RP105 gene revealed impaired proliferative and humoral immune responses of RP105-deficient B cells to LPS. Using originally LPS-unresponsive Ba/F3 cells expressing exogenous TLR4 and RP105, we demonstrate the functional cooperation between TLR4 and RP105 in LPS-induced nuclear factor .kappa.B activation. These data suggest the existence of the TLR4-RP105 signaling module in the LPS-induced B cell activation.
IT Proteins, specific or class
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**RP-105**; Toll-like receptor protein RP105 regulates lipopolysaccharide signaling in B cells)

L2 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2002 ACS
TI Roles of toll-like receptor (TLR) family in innate immunity
SO Mol. Med. (Tokyo) (1999), 36(5), 488-497

CODEN: MOLMEL; ISSN: 0918-6557

AB A review with 24 refs. on structures and functions of LRR (leucine-rich repeat), TLR (Toll-like receptor), and **RP 105** on mechanisms of TLR signal transduction, on roles of TLR in sensitivity to lipopolysaccharide, and on roles of TLR in autoimmune diseases.

AB A review with 24 refs. on structures and functions of LRR (leucine-rich repeat), TLR (Toll-like receptor), and **RP 105** on mechanisms of TLR signal transduction, on roles of TLR in sensitivity to lipopolysaccharide, and on roles of TLR in. . .

L2 ANSWER 6 OF 10 MEDLINE DUPLICATE 2

TI Human MD-1 homologue is a BCG-regulated gene product in monocytes: its identification by differential display.

SO BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1999 Mar 16) 256 (2) 325-9.
Journal code: 9Y8; 0372516. ISSN: 0006-291X.

AB BCG-CWS is a therapeutically potent immune activator which improves the prognosis of cancer patients. However, the targeting effector cells and molecules for BCG-CWS in the human immune system have not been determined.
Here, we found that BCG-CWS activates human monocytes and concomitantly down-regulates expression of a human homologue of chicken MD-1 in the activated monocytes by differential display. According to a previous study, MD-1 forms a complex with the Toll family protein **RP-105** on murine B cell lines to facilitate its stable expression. Thus, MD-1 may participate in regulation of innate immune activation on human monocytes. Our results, taken together with these recent findings regarding Toll family proteins, suggest that BCG-CWS acts on monocytes to modulate the human innate immune system via regulation of Toll family proteins.

AB . . . the activated monocytes by differential display. According to a previous study, MD-1 forms a complex with the Toll family protein **RP-105** on murine B cell lines to facilitate its stable expression. Thus, MD-1 may participate in regulation of innate immune activation. . .

L2 ANSWER 7 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

TI DNA sequence of human **RP-105**.

SO Official Gazette of the United States Patent and Trademark Office Patents, (July 14, 1998) Vol. 1212, No. 2, pp. 1877.
ISSN: 0098-1133.

TI DNA sequence of human **RP-105**.

IT Miscellaneous Descriptors
ATCC DEPOSIT NO 69902; BIOTECHNOLOGY; CDNA SEQUENCE; CLONE; HUMAN **RP-105**

L2 ANSWER 8 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

TI RP105 is associated with MD-1 and transmits an activation signal in human B cells.

SO Blood, (Oct. 15, 1998) Vol. 92, No. 8, pp. 2815-2822.
ISSN: 0006-4971.

AB RP105 was originally discovered as a mouse B-cell surface molecule that transmits an activation signal. The signal leads to resistance against irradiation-induced apoptosis and massive B-cell proliferation. Recently, we found that mouse RP105 is associated with another molecule, MD-1. We

have isolated here the human MD-1 cDNA. We show that human MD-1 is also associated with human RP105 and has an important role in call surface expression of RP105. We also describe a monoclonal antibody (MoAb) that recognizes human RP105. Expression of RP105 is restricted to CD19+ B cells. Histological studies showed that RP105 is expressed mainly on mature B cells in mantle zones. Germinal center calls are either dull or negative. RP105 is thus a novel human B-cell marker that is preferentially expressed on mature B cells. Moreover, the anti-RP105 MoAb activates B cells, leading to increases in cell size, expression of a costimulatory molecule CD80, and DNA synthesis. The B-cell activation pathway using RP105 is conserved in humans.

IT . . .
(Transport and Circulation); Molecular Genetics (Biochemistry and Molecular Biophysics)

IT Chemicals & Biochemicals
MD-1 protein: amino acid sequence, nucleotide sequence; **RP-105** leucine-rich repeat containing molecule: B-cell activation signal transmitter, MD-1 protein association

L2 ANSWER 9 OF 10 MEDLINE DUPLICATE 3

TI The molecular mechanism of B cell activation by toll-like receptor protein **RP-105**.

SO JOURNAL OF EXPERIMENTAL MEDICINE, (1998 Jul 6) 188 (1) 93-101. Journal code: I2V; 2985109R. ISSN: 0022-1007.

AB The B cell-specific transmembrane protein **RP-105** belongs to the family of Drosophila toll-like proteins which are likely to trigger innate immune responses in mice and man. Here we demonstrate that the Src-family protein tyrosine kinase Lyn, protein kinase C beta I/II (PKCbetaI/II), and Erk2-specific mitogen-activated protein (MAP) kinase kinase (MEK) are essential and probably functionally connected elements of the **RP-105**-mediated signaling cascade in B cells. We also find that negative regulation of **RP-105**-mediated activation of MAP kinases by membrane immunoglobulin may account for the phenomenon of antigen receptor-mediated arrest of **RP-105** -mediated B cell proliferation.

TI The molecular mechanism of B cell activation by toll-like receptor protein **RP-105**.

AB The B cell-specific transmembrane protein **RP-105** belongs to the family of Drosophila toll-like proteins which are likely to trigger innate immune responses in mice and man.. . . beta I/II (PKCbetaI/II), and Erk2-specific mitogen-activated protein (MAP) kinase kinase (MEK) are essential and probably functionally connected elements of the **RP-105**-mediated signaling cascade in B cells. We also find that negative regulation of **RP-105**-mediated activation of MAP kinases by membrane immunoglobulin may account for the phenomenon of antigen receptor-mediated arrest of **RP-105** -mediated B cell proliferation.

L2 ANSWER 10 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

TI Lack of expression of a human RP105 homologue on B cells in SLE.

SO Arthritis & Rheumatism, (Sept., 1998) Vol. 41, No. 9 SUPPL., pp. S74.

Meeting Info.: 62nd National Scientific Meeting of the American College of Rheumatology and the 33rd National Scientific Meeting of the Association of Rheumatology Health Professionals San Diego, California, USA November 8-12, 1998 American College of Rheumatology
. ISSN: 0004-3591.

IT . . .
Immunology (Human Medicine, Medical Sciences)

IT Diseases
systemic lupus erythematosus: connective tissue disease, immune system disease

IT Chemicals & Biochemicals
RP-105 surface protein: negative B-cell expression

=>